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Short Communication

Disentangling two underlying processes in the initial phase of substance use: Onset and frequency of use in adolescent smoking

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ABSTRACT

Purpose: Most studies on adolescent smoking focus either on the probability of smoking onset or frequency of smoking. We assume the existence of two different qualitatively distinct processes in smoking acquisition. Therefore our objective was to test a two-part latent growth model, which assumes that psychosocial variables associated with the probability of smoking onset are different from, or differently related to variables associated with frequency of smoking given smoking onset.
Methods: The predictive associations of blocks of variables of (1) intrapersonal factors, (2) cognitions, (3) role models, and (4) family variables, on both smoking onset, and frequency of smoking *given* smoking onset, were tested in a nationwide sample of Dutch adolescents by using a two-part model.
Summary: Smoking onset was instigated by a variety of factors, while similar and other factors predicted frequency of smoking given smoking onset itself. Self-esteem, attitudes, and proportion of friends smoking, were identified as factors that affected both absolute smoking and frequency of smoking.
Overall conclusions: This study illustrates that it makes sense to differentiate between smoking onset and frequency of smoking and that few factors are active in both processes.

1. Introduction

The preponderance of existing smoking prevention programs is not effective or only marginal (Glantz & Mandel, 2005). One reason may be that these programs partially ignore that the process towards regular smoking is roughly comprised of two distinct processes. This is in line with studies finding a semi continuous response on smoking: a large number of adolescents who do not smoke at all and a variety of smoking frequencies among those who *do* report smoking. In the present study we will distinguish smoking onset from smoking frequency by using a two-part model (Olsen & Schafer, 2001). A two-part model decomposes the original distribution of tobacco use into one a part that describes the probability of onset and a second part that analyses the frequency of use *given* onset. Decomposing the distribution of tobacco use and integrating both parts in one model may help to better understand the process of adolescent smoking acquisition. We will systematically assess the predictive associations of the comprehensive overview of theoretical domains suggested in the review by Petraitis, Flay, and Miller (1995) while this review offers a comprehensive overview of factors from different theories.

2. Methodology

2.1. Participants and procedure

Data were from three waves (one-year intervals) of a longitudinal study on adolescent smoking (Otten, Engels, & Van den Eijnden, 2005). Students of first and second year classes of 33 secondary schools participated. Participants needed to have data on each predictor variable to be included in the analyses, leading to 2565 participants (73%). At T1, mean age was 13.14 ($SD = 0.70$), 54% of the students were female.

2.2. Measures

2.2.1. Adolescent smoking

Respondents indicated their smoking behaviour on a 7-point scale (*0 cigarettes per day*, *<1 cigarette per day*, *1–5 cigarettes per day*, *6–10 cigarettes per day*, *11–20 cigarettes per day*, *21–30 cigarettes per day*, and *≥31 cigarettes per day*). The probability of smoking onset was separated from the rest of the distribution by creating binary indicator variables distinguishing cases that reported having used tobacco (coded 1), from youths who reported non-use (coded 0). Respondents with scores above 0 ended up in the second part of the model assessing smoking frequency.

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2.2.2. Predictor variables

2.2.2.1. Intrapersonal characteristics. We measured the personality dimensions *extraversion and emotional instability* (Quick Big Five; Vermulst, 2005), *depressive feelings* (Depressive Mood List; Kandel & Davies, 1986), *self-esteem* (Rosenberg's Self-Esteem scale, 1965) and *loneliness* (LLCA; Marcoen, Goossens, & Caes, 1987).

2.2.2.2. Substance-specific cognitions. We measured *attitudes* toward daily smoking (Dijkstra, De Vries, & Bakker, 1996) and *self-efficacy* to refrain from smoking in tempting situations (Engels, Knibbe, De Vries, & Drop, 1998).

2.2.2.3. Substance-using role models. We assessed *parental smoking* by combining smoking status of both parents (1 = both parents are non-smokers (60.7%), 2 = one parent is a smoker (24.6%), 3 = both parents are smokers (14.7%)) (Farkas, Distefan, Choi, Gilpin, & Pierce, 1999), *best friends' smoking* by asking respondents to write down whether his/her best friend was a smoker (92.3%) or a non-smoker (7.7%) (Engels, Knibbe, De Vries, Drop, & Van Breukelen, 1999), and the *proportion of smoking friends* (1 = no smoking friends (67.4%), 2 = less than 50% of my friends smoke (26.1%), and 3 = 50% or more of my friends smoke (6.4%)).

2.2.2.4. Family factors. Parental highest education was assessed by asking respondents to report the highest level of education completed by both parents (lower (29.5%), intermediate (general) (43.4%) and high education level (senior secondary and pre-university education) (27.1%)). Regarding *neglectful parenting*, we assessed parenting dimensions of involvement and strictness (Steinberg, Fletcher, & Darling, 1994). Neglectful parents were those parents who scored in the lower parts of both dimensions (N = 789). Finally, we assessed *family constellations* (families with both parents (90.8%) and single-parent families).[1]

2.3. Statistical approach

We used a two-part latent growth model (Olsen & Schafer, 2001), which is a longitudinal adaptation of a two-equation model or zero-inflated model (Atkins & Gallop, 2007), used to model data with a preponderance of zero observations on the outcome variable. A two-part model assumes that variables associated with the probability of engaging in a type of behaviour differ from variables associated with the frequency of that behaviour, given that some engagement is present. (For the stochastic specification, Brown, Catalano, Fleming, Haggerty, & Abbott, 2005.)

The two-part model decomposes the original distribution of tobacco use outcome into two parts, each modeled by separate, but correlated growth functions (see Fig. 1). In part 1, the probability of smoking onset was separated from the rest of the distribution by creating binary indicator variables distinguishing cases that reported having used tobacco (scores of 1 or higher; coded 1), from youths who reported non-use (coded 0). The resulting annually assessed smoking onset indicators were modeled through a random effects logistic regression model in which the log odds of use were regressed on growth parameters. Thus, in this part, the probability of smoking onset with age was captured by latent growth parameters, where the intercept refers to initial level differences (i.e., probability of use at age 13), and the linear slope to change in probability of use over time (i.e., increase or decrease in probability of use with age). In part 2 of the model, the continuous indicators of the resulting frequency of tobacco use were used, given onset. So, in part 2 of the model only the youths who reported having used tobacco were included. We then included the predictor variables. Because we estimated the growth parameters in the two-part model simultaneously, it enabled us to control for the conditionality of the frequency-of-use outcome on the initial decision to engage in smoking when estimating the relationships between growth factors and covariates.

[1] All measures have been used frequently and have good psychometric qualities.

3. Results

We first determined the number of growth parameters for parts 1 and 2 of the model separately. In both parts, models with intercepts and slopes were superior to intercept-only models. The model for part 1 had significant variances on both the intercept (variance = 5.30, SE = 1.23, $p<.01$) and the slope parameters (variance = 3.08, SE = 0.82, $p<.01$). For part 2, no significant covariance between the level (intercept) and slope parameter was found ($B=-.001$, SE = 0.02, $p>.05$) and was therefore held equal to zero for the remainder of the analyses. Both parameters had a significant variance: intercept, variance = 0.11, SE = 0.02, $p<.01$; slope, variance = 0.06, SE = 0.01, $p<.01$). Part 2 of the model had a good fit to the data: CFI = .98, TLI = .98, RMSEA = .05. We then specified the two-part model, correlating growth parameters of both parts.[2]

In Table 1a, results for part 1 of the model are expressed as odds ratios. Only associations with the levels of the probability of use (intercept) were found. Children with a positive attitude toward smoking, and with friends or a best friend who smoked were more likely to also smoke. Higher levels of self-esteem and self-efficacy protected children from smoking onset.

The associations of the covariates with level and growth in frequency, given onset are in Table 1b. Associations with both the level of use (intercept) and growth in use (slope) were found. Boys showed stronger increases in smoking frequency than girls. Older respondents were more likely to have higher initial levels of smoking frequency. Younger respondents on the other hand, were more likely to show stronger increases in smoking frequency than older respondents. Regarding intrapersonal factors, the only significant effect was found for self-esteem: respondents with higher levels of self-esteem showed lower initial levels of smoking frequency. Regarding cognitive factors, youths who had a more positive attitude toward smoking showed a stronger increase in levels of smoking frequency. Youths whose parents smoked or who had many friends who smoked also had higher smoking frequencies at age 13. Finally, respondents who experienced a neglectful parenting style were likely to engage in more frequent smoking with age. Self-esteem and having friends who smoke had effects on both smoking onset and frequency. We did not find support for any moderation.

4. Discussion

Our objective was to account for the robust distinction between two processes by exploring whether different predictors discriminate between smoking onset and frequency, by using a two-part model. Smoking onset was determined by a variety of factors. Smoking frequency, given smoking onset, was related with gender, age, self-esteem, attitudes, exposure to smoking parents and friends, and neglectful parenting. Proportion of smoking friends and self-esteem had an effect on both onset *and* frequency.

Regarding intrapersonal factors, self-esteem appeared to be important. It has been suggested that the relationship between self-esteem and smoking differs in groups that vary in terms of peer-orientation. Glendinning and Inglis (1999) found that the group in which the focus was on spending time with friends and holding largely negative attitudes toward authority and control, reported low levels of self-esteem and high smoking rates. Other intrapersonal

[2] Descriptives and results of the different analytic steps can be provided upon request by the first author.

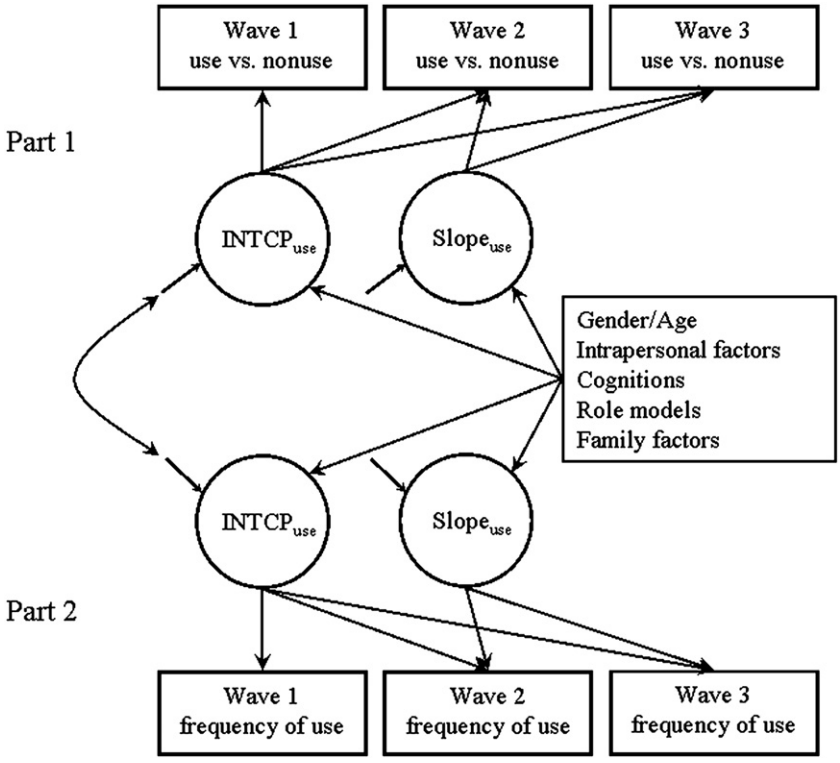

Fig. 1. Two-part model predicting smoking onset vs. non-use (Part 1) and frequency of use, given smoking onset (Part 2) by intrapersonal factors, cognitions, role models, and family risk factors.

traits than self-esteem were not significant in either parts of the model, which may be explained by the idea that some factors become more important later in life.

A pro-smoking attitude was associated with the probability of smoking onset and frequency. While attitudes may predict smoking onset, this might be harder to conclude from increasing frequency since a pro-smoking attitude might be a by-product of increasing frequency of smoking itself. Self-efficacy was only significant in the model's first part, supporting our initial idea that self-efficacy is particularly important in onset.

In both smoking onset and smoking frequency the proportion of smoking peers was a precursor. In smoking onset the proportion of friends smoking can be expected to mainly exert its influence through mechanisms of social learning (Bandura, 1977). In smoking frequency however, other processes may be active that may have caused a significant effect. Best friends smoking was only related to smoking onset, suggesting that it is the mere presence of people who smoke, rather than relationship quality.

Parental smoking (as well as neglectful parenting) was only significant for smoking frequency. The effects of parental smoking in

Table 1a
Part 1 of the Two-part model: factors discriminating use vs. non-use (smoking onset).

Part 1: Smoking onset				
	Intercept		Slope	
	OR	95% CI	OR	95% CI
Sex	0.97	0.62–1.52	0.86	0.67–1.10
Age	0.97	0.81–1.16	0.87	0.72–1.06
Intrapersonal				
Extraversion	1.28	0.95–1.72	1.22	0.97–1.53
Emotional Instability	0.90	0.67–1.21	0.94	0.80–1.12
Depression	1.15	0.52–2.55	1.14	0.76–1.69
Loneliness	1.22	0.91–1.65	0.85	0.60–1.19
Self-esteem	0.49	0.37–0.65**	0.98	0.63–1.55
Cognitive				
Attitude	2.11	1.64–2.73**	1.49	0.81–2.74
Self-efficacy	0.52	0.33–0.82**	0.94	0.67–1.32
Role models				
Parents smoking	1.28	0.74–2.21	1.19	0.89–1.58
Prop. friends smoking	2.95	1.84–4.73**	1.03	0.57–1.86
Best friend smokes	4.08	1.34–12.46*	0.61	0.19–2.01
Family				
Household SES	1.06	0.78–1.44	0.87	0.71–1.06
Neglectful parenting	1.09	0.47–2.51	1.25	0.83–1.88

Note. $^{**}p<.01$, $^{*}p<.05$.

Table 1b
Part 2 of the two-part model: factors associated with frequency of smoking.

Part 2: Smoking frequency								
	Intercept				Slope			
	B	*SE*	β	*r*	B	*SE*	β	*r*
Sex	0.01	0.04	0.01	.10	−0.10	0.03	−0.17**	.41
Age	0.11	0.03	0.22**	.47	−0.09	0.02	−0.22**	.47
Intrapersonal								
Extraversion	0.01	0.03	0.02	.14	0.01	0.02	0.05	.22
Emotional Instability	0.01	0.03	0.01	.01	−0.02	0.02	−0.09	.30
Depression	−0.07	0.04	−0.13	.36	0.04	0.03	0.08	.28
Loneliness	0.03	0.05	0.05	.22	−0.04	0.04	−0.08	.28
Self-esteem	−0.14	0.07	−0.22*	.47	0.05	0.04	0.10	.32
Cognitive								
Attitude	0.02	0.04	0.05	.22	0.06	0.03	0.19*	.44
Self-efficacy	−0.07	0.05	−0.13	.36	−0.01	0.03	−0.02	.14
Role models								
Parents smoking	0.10	0.03	0.20**	.45	0.01	0.02	0.02	.14
Prop. friends smoking	0.12	0.04	0.24**	.49	0.01	0.03	0.02	.14
Best friend smokes	0.08	0.09	0.06	.24	−0.00	0.04	−0.00	.00
Family								
Household SES	−0.04	0.02	−0.17	.41	−0.00	0.01	−0.02	.14
Neglectful parenting	0.04	0.06	0.05	.22	0.06	0.02	0.09**	.30

Note. $^{**}p<.01$, $^{*}p<.05$, r's represent multiple correlation coefficients indicating effect size.

distinguishing smoking onset and non-use however may be indirectly exerting its influence through attitudes (Otten, Harakeh, Vermulst, Van den Eijnden, & Engels, 2007). The direct effects of parental smoking on frequency of smoking may be due to increased parental tolerance, and the availability of cigarettes (e.g., Jackson & Henriksen, 1997). Neglectful parenting was associated with increases in smoking frequency but not with onset. Increasing smoking frequency may be indicative for development of a smoking addiction, for which inadequate parenting has been considered a childhood factor (Moffitt & Caspi, 2001).

Finally, all participants were about the same age; however older adolescents were likely to smoke more than younger adolescents, whereas younger adolescents were more likely to increase in smoking frequency. The latter may indicate that younger adolescents are likely to model their older peers who already have increased the level of smoking. Although there were no differences in levels of smoking frequency, boys were more likely to increase in frequency than girls, which is surprising while girls have been found to develop a nicotine addiction faster than boys (Warren, Jones, Eriksen, & Asma, 2006). However, it might be that some boys are more extreme in their behaviour once they started while girls are generally more likely to develop a nicotine addiction without progressing to extreme levels of use.

5. Conclusions

Our results showed that common and process-specific predictors are associated with smoking onset and frequency. Not distinguishing between the different processes at work could preclude our understanding of youths' smoking behaviours. Moreover, not distinguishing between these processes in the early stages of adolescent smoking acquisition could easily lead to inaccurate expectations from prevention campaigns and interventions aimed at preventing adolescent smoking or reducing young adolescent smoking rates.

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Contributors

Rutger Engels and Roy Otten designed the study. Roy Otten wrote the manuscript and Pol van Lier conducted the analyses. All authors contributed to and have approved the final manuscript.

Conflict of Interest

None.

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